WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences

Search Results -

Terms	Documents
16 same patient$	0

Database:
- US Patents Full-Text Database
- US Pre-Grant Publication Full-Text Database
- JPO Abstracts Database
- EPO Abstracts Database
- Derwent World Patents Index
- IBM Technical Disclosure Bulletins

Refine Search: `16 same patient$` Clear

Search History

Today's Date: 5/16/2001

DB Name	Query	Hit Count	Set Name
USPT	16 same patient$	0	L10
USPT	15 same patient$	0	L9
USPT	16 same treat$	3	L8
USPT	15 same treat$	0	L7
USPT	14 same (muta$ or varian$)	21	L6
USPT	13 same (muta$ or varian$)	8	L5
USPT	12 same gene$	97	L4
USPT	11 same gene$	41	L3
USPT	pyrimidine same (transport$ or metaboli$)	602	L2
USPT	folate same (transport$ or metaboli$)	308	L1

L8: Entry 2 of 3 File: USPT Jan 5, 1999

DOCUMENT-IDENTIFIER: US 5856153 A
TITLE: Suicide genes and new associations of pyrimidine nucleobase and nucleoside analogs with new suicide genes for gene therapy of acquired diseases

BSPR:
The codA gene of Escherichia coli encoding cytosine deaminase (hereinafter referred to as CDase) represents another potential suicide gene to be used for the selective elimination of unwanted human cells. Cytosine deaminase is the first enzyme of the only metabolic pathway by which exogeneous cytosine or endogeneous cytosine from pyrimidine nucleotide breakdown is utilized by way of hydrolytic deamination to uracil and ammonia. Cytosine deaminases have been found in prokaryotes and lower eukaryotes but appear to be absent in higher eukaryotes, both in mammals as well as in plants [Koechlin et al., Biochem Parmacol. 15, 435-446 (1966)] [Ross, C. Plant Physiol. 40, 65-73 (1965)]. Cytosine deaminase also deaminates the innocuous fluorocytosine (hereinafter referred to as FC) into fluorouracil (hereinafter referred to as FU), a highly toxic compound when efficiently converted to 5-fluoro-UMP. Cells lacking cytosine deaminase activity either as a consequence of a mutational inactivation as illustrated by codA and fcyl (genes coding for cytosine deaminase) mutants of Escherichia. coli and Saccharomyces cerevisiae respectively, or because they are naturally deficient for this enzyme, as are mammalian and plant cells, are resistant to 5-fluorocytosine [Kilstrup et al., J. Bacteriol 171, 2124-7 (1989)] [Jund R. & Lacroute F. Journal of Bacteriology 102, 607-615 (1970)]. This property provides the basis for the use of the E.coli coda gene as a suicide or a negative selection gene in a number of recently reported experiments with mammalian and plant cells [Huber et al., Cancer Res 53, 4619-26 (1993); WO 93/01281; Mullen et al., Cancer Res 54, 1503-6 (1994)] where transformed cells were shown to have acquired cytosine deaminase activity and to be sensitive to treatment with 5-fluorocytosine.

BSPR:
The nucleotide dTTP is an essential metabolite, finely regulated in all cells. A severe depletion of dTTP by inhibitors or mutations induces the thymineless death of cells, possibly by induction of an endonuclease responsible for DNA double stranded breaks. The use of FC, activated by a gene coding for CDase and UPRTase activities, is intended to kill targeted cells by thymidine death due to dTTP starvation. Thymidine relieves the toxic effect of FC by furnishing dTTP after bypassing the thymidylate synthase blockage through the pyrimidine salvage pathway (see FIG. 1). The use of thymidine or deoxyuridine analogs in conjunction with FC for treating cells expressing two activator suicide genes has the opposite effect. The incorporation of triphosphate analogs into DNA by the replication and repair processes is facilitated by lack of the competing dTTP, leading to a more pronounced killing effect.

L8: Entry 1 of 3 File: USPT Mar 30, 1999

DOCUMENT-IDENTIFIER: US 5888799 A
TITLE: Recombinant avirulent bacterial antigen delivery system

DEPR:
DNA molecules from S. mutans strains 6715 (serotype g, 45% guanine+cytosine content) and PS14 and GS-5 (serotype c, 35% guanine+cytosine) were cloned into suitable strains of E. coli K-12. Shotgun cloning experiments were performed to determine whether S. mutans genes were expressed in E. coli and, if so, whether they would complement E. coli gene defects. The DNA was isolated from the S. mutans strains UAB50 (PS14), UAB90, (PS-14), and UAB308 (GS-5) by treating the S. mutans cells with the enzyme mutanolysin and then lysing the bacteria with the detergent sodium dodecyl sulfate. The DNA was recovered by ethanol precipitation, restricted with various restriction endonucleases such as EcoRI, HindIII, BamHI, and PstI and used to anneal to pBR322 or pACYC184 vectors cut with the homologous enzyme. Recombinant molecules were formed by the addition of polynucleotide joining enzyme (or DNA ligase) and suitable strains of E. coli K-12 such as HB101, .chi. 1274, and .chi. 1849 were transformed by the calcium chloride cold shock method. In other experiments, recombinant molecules were formed by ligating DNA to the cosmid vector pJC74, packaging the recombinant molecules by in vitro packaging methodology with components to introduce the recombinant cosmid DNA into suitable strains of E. coli K-12 such as HB101 lysogenic for the thermo-inducible lambda prophage .chi. cI857. Transformant or transductant clones were selected for resistance to an antibiotic for which the cloning vector carried the appropriate drug resistance gene. Tests using a variety of multiple mutant E. coli strains indicated that about 40% of the tested E. coli gene defects for purine, pyrimidine and amino acid biosynthesis and carbohydrate use could be complemented by S. mutans genetic information. The presence of S. mutans DNA was verified using Southern blotting analysis. E. coli deletion mutants lacking a given function would sometimes grow as rapidly with S. mutans genetic information cloned on the multicopy plasmids pBR322 and pACYC184 (available commerically from Bethesda Research Laboratory, Rockville, Md.) as they would if provided with the optimal amount of the required supplement. In general, most S. mutans genes were expressed constitutively and were not subject to repression by end products or inducible by appropriate substrates. Gene products of S. mutans that are necessary for the transport and phosphorylation of sugars and that probably associate with the cytoplasmic membrance of S. mutans function in E. coli in much the same way. S. mutans gene products that are normally on the cell surface of S. mutans or are excreted into the growth medium were is transported across the E. coli cytoplasmic membrane and ended up in the periplasmic space. E. coli perA mutants that are defective in transport of various periplasmic proteins from the cytoplasm into the periplasm were still able to transport certain S. mutans cell surface gene products into the periplasmic space in E. coli.